

## CARDIOVASCULAR MEDICINE

# Can coronary flow velocity reserve determined by transthoracic Doppler echocardiography predict the recovery of regional left ventricular function in patients with acute myocardial infarction?

Y Ueno, Y Nakamura, M Kinoshita, T Fujita, T Sakamoto, H Okamura

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See end of article for authors' affiliations

Correspondence to:  
Dr Yoshiki Ueno, The First  
Department of Internal  
Medicine, Shiga University  
of Medical Science,  
Tsukinowa, Otsu, Shiga,  
520-2192, Japan;  
uenoy@belle.shiga-med.ac.jp

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**Objective:** To determine whether the early assessment of coronary flow velocity reserve (CFVR) by transthoracic Doppler echocardiography (TTDE) can predict myocardial viability after revascularisation in patients with acute myocardial infarction.

**Methods:** 29 patients with anterior acute myocardial infarction who were successfully treated by coronary angioplasty were studied. TTDE was used to record coronary flow velocities in the distal left anterior descending artery at rest and during hyperaemia induced by intravenous infusion of adenosine triphosphate. CFVR was calculated immediately and 24 hours after revascularisation and at discharge. Regional wall motion was analysed to calculate the anterior wall motion score index (A-WMSI) by two dimensional echocardiography before revascularisation and at discharge.

**Results:** CFVR immediately and 24 hours after revascularisation correlated significantly with A-WMSI at discharge ( $r = -0.58$ ,  $p < 0.001$  and  $r = -0.80$ ,  $p < 0.0001$ , respectively). CFVR 24 hours after revascularisation was a better predictor of recovery of regional left ventricular function than CFVR immediately after revascularisation. The optimal cut off ratio for predicting viable myocardium was 1.5 for CFVR 24 hours after revascularisation (sensitivity = 94%, specificity = 91%).

**Conclusions:** CFVR by TTDE was useful for predicting the recovery of left ventricular function after revascularisation in patients with acute myocardial infarction

Revascularisation in patients with acute myocardial infarction enables the early restoration of adequate reflow in the infarct related coronary artery. However, patency of the infarct related coronary artery does not always guarantee salvage of myocardium at risk of ischaemia. Even without significant epicardial coronary stenosis, with the use of myocardial contrast echocardiography we have sometimes recognised the absence of blood flow—that is, “no reflow”—resulting from microvascular damage.<sup>1–3</sup> Previous studies have suggested that coronary flow velocity reserve (CFVR) measured by invasive methods can be used to evaluate microvascular function and may help to assess myocardial viability and consequently predict further left ventricular wall motion recovery in patients with acute myocardial infarction.<sup>4</sup>

Transthoracic Doppler echocardiography (TTDE) is widely used in the clinical setting and makes it possible to measure CFVR in the left anterior descending coronary artery.<sup>5,6</sup> However, the relation between CFVR by TTDE and the recovery of left ventricular function has not been evaluated. We designed this study to determine whether CFVR as determined by TTDE can be used to predict left ventricular wall motion recovery in patients with acute myocardial infarction.

## METHODS

### Patient population and study

The study population consisted of 35 consecutive patients who had undergone successful recanalisation at their first acute anterior myocardial infarction by primary coronary angioplasty within 12 hours after the onset of chest pain. The diagnosis of acute myocardial infarction was based on chest pain lasting > 30 minutes, ST segment elevation > 2.0 mm in at least two contiguous ECG leads, an increase in serum creatine

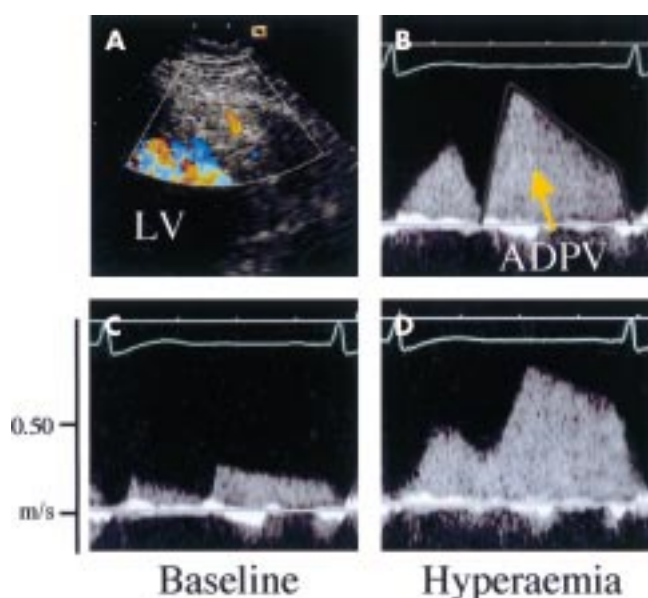
kinase to more than threefold the normal value, and TIMI (thrombolysis in myocardial infarction) grade 0, 1, or 2 flow at the initial coronary angiography.<sup>7</sup> We excluded patients with a cardiac event during follow up, atrial fibrillation, significant valvar heart disease, an inadequate recording of the coronary flow velocity spectrum, or a poor echocardiographic image. The remaining 29 patients (18 men, 11 women, mean (SD) age 61 (13) years) were enrolled in this study.

Regional wall motion was analysed by two dimensional echocardiography before revascularisation and at discharge (20 (4) days after acute myocardial infarction). CFVR was measured immediately and 24 hours after revascularisation and at discharge. The study protocol was approved by the ethics committee of Shiga University of Medical Science. All of the patients gave their informed consent before cardiac catheterisation.

### Coronary angiography and angioplasty

Before angiography, all of the patients received an intravenous bolus injection of 5000 U of heparin and an intracoronary bolus injection of 2 mg of isosorbide dinitrate. Diagnostic coronary angiography was performed using the Judkins technique. After an additional intravenous bolus injection of 5000 U of heparin, coronary angioplasty was performed by standard techniques and rescue stenting was performed if necessary. Procedural success was defined as residual stenosis of < 25%.

**Abbreviations:** A-WMSI, anterior wall motion score index; CFVR, coronary flow velocity reserve; TIMI, thrombolysis in myocardial infarction; TTDE transthoracic Doppler echocardiography



**Figure 1** Transthoracic Doppler echocardiogram showing coronary blood flow and coronary flow velocity recordings in the distal left anterior descending artery. (A) Colour Doppler flow mapping. (B) Coronary flow velocity recording. ADPV, average diastolic peak velocity. Example of coronary flow velocity recordings (C) at baseline and (D) during hyperaemia.

Patients received conventional drug treatment according to their individual needs, which were determined by the attending physician. Each patient underwent predischARGE coronary angiography 18 (3) days after acute myocardial infarction.

### Angiographic data analysis

Images obtained by coronary angiography were stored on compact disks for off line analysis (Cardiovascular Measurement System, Medical Imaging System, Leiden, Netherlands).<sup>8</sup> Contrast flow through the infarct related coronary artery was graded by means of the TIMI flow classification.<sup>7</sup> Collateral flow was graded according to the Rentrop classification based on the initial coronary angiogram.<sup>9</sup>

### CFVR measurement by TTDE

Echocardiographic examinations were performed with a Sequoia C256 (Acuson, Mountain View, California, USA) digital ultrasonographic system with a 3.5–7 MHz transducer. In

colour Doppler flow mapping, the velocity range was set at  $\pm 12.0$  or  $\pm 16.0$  cm/s. The colour gain was adjusted to provide optimal imaging. The acoustic window was around the midclavicular line in the fourth or fifth intercostal space in the left lateral decubitus position. First the ventricle was imaged in the long axis cross section and the ultrasound beam was inclined laterally. Next, coronary blood flow in the distal portion of the left anterior descending coronary artery was visualised by 5 MHz colour Doppler. Coronary flow velocity was measured with pulsed wave Doppler at minimum angle correction. Angle correction was needed in each examination because of the incident Doppler angle (mean angle  $28^\circ$ , range  $15$ – $44^\circ$ ). Stop frames and clips were digitally recorded on magneto-optical disks.

The coronary blood flow velocity profile at the distal part of the left anterior descending coronary artery was biphasic. The average diastolic peak velocity was measured by manually tracing the Doppler spectrum with an analysis system that was incorporated into the ultrasonographic system. Since it was difficult to obtain complete Doppler spectral envelopes throughout the cardiac cycle as a result of cardiac motion, only the average diastolic peak velocity was measured. CFVR was defined as the ratio of the hyperaemic average diastolic peak velocity (induced by the intravenous infusion of  $0.15$  mg/kg/min adenosine triphosphate) to the baseline average diastolic peak velocity (fig 1). Adenosine triphosphate was administered for two minutes to record spectral Doppler signals under hyperaemic conditions.

The final value for the flow velocity is the average of five cardiac cycles. All patients had continuous heart rate and ECG monitoring. Blood pressure was recorded at baseline, every minute during adenosine triphosphate infusion, and at recovery.

### Regional wall motion analysis

Left ventricular wall motion was analysed according to the 16 segment model of the American Society of Echocardiography.<sup>10</sup> Nine of the 16 segments were determined to be in the vicinity of the left anterior descending coronary artery and the anterior wall motion score index (A-WMSI) was calculated as an average of the wall motion scores in these nine segments. Stop frames and clips were digitally recorded on magneto-optical disks for off line analysis. An observer who had no knowledge of other patient data calculated A-WMSI from two dimensional echocardiography.

### Statistical analysis

Continuous variables are expressed as the mean (SD) and were compared by Student's *t* test. Categorical variables were

**Table 1** Baseline characteristics in the viable myocardium and non-viable myocardium groups

	Viable myocardium (n=18)	Non-viable myocardium (n=11)	p Value
Age (years)	60 (13)	62 (13)	NS
Male sex (%)	11 (61)	7 (64)	NS
Reperfusion time (min)	330 (209)	328 (227)	NS
Peak CK (IU/l)	2954 (1533)	5017 (2270)	0.007
Q wave infarction (%)	15 (83)	11 (100)	NS
Hypertension (%)	8 (44)	4 (36)	NS
Diabetes mellitus (%)	3 (16)	4 (36)	NS
TC >5.7 mmol/l (%)	5 (28)	3 (27)	NS
Smoking (%)	9 (50)	6 (55)	NS
Culprit lesion proximal LAD (%)	9 (50)	7 (64)	NS
Stent (%)	16 (89)	10 (91)	NS
MVD (%)	5 (28)	6 (55)	NS
Rentrop grade 0 or 1 (%)	12 (67)	10 (91)	NS
TIMI 3 reflow (%)	18 (100)	8 (73)	0.02
DS before revascularisation (%)	97 (5)	99 (3)	NS
DS after revascularisation (%)	7 (7)	6 (6)	NS

Data are mean (SD) or n (%)

CK, creatine kinase; DS, diameter stenosis; LAD, left anterior descending coronary artery; MVD, multivessel disease; TC, total cholesterol; TIMI, thrombolysis in myocardial infarction.

**Table 2** Echocardiographic results in the viable myocardium and non-viable myocardium groups

	Viable myocardium (n=18)	Non-viable myocardium (n=11)	p Value
A-WMSI before revascularisation	2.57 (0.18)	2.63 (0.11)	NS
A-WMSI at discharge	1.62 (0.22)*	2.43 (0.19)†	<0.0001
CFVR immediately after revascularisation	1.46 (0.07)	1.36 (0.09)	0.003
CFVR 24 hours after revascularisation	1.71 (0.26)‡	1.30 (0.14)	<0.0001
CFVR at discharge	2.25 (0.43)‡§	1.94 (0.23)‡§	0.04

\*p<0.0001 v before revascularisation; †p<0.05 v before revascularisation; ‡p<0.0001 v immediately after revascularisation; §p<0.0001 v 24 hours after revascularisation. A-WMSI, anterior wall motion score index; CFVR, coronary flow velocity reserve.

**Table 3** Univariate regression analysis between CFVR and A-WMSI at discharge

	r	p Value
CFVR immediately after revascularisation	-0.58	0.001
CFVR 24 hours after revascularisation	-0.80	<0.0001
CFVR at discharge	-0.50	0.006

compared by the  $\chi^2$  test. Linear regression analysis was used to estimate the relation between CFVR and A-WMSI at discharge. Differences were considered significant at  $p < 0.05$ . To test interobserver and intraobserver variability, two independent observers measured CFVR in 20 randomly selected patients.

## RESULTS

### Patient characteristics and clinical results

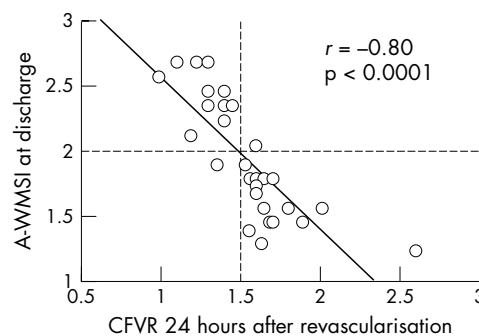
The mean duration from the onset of symptoms to coronary reperfusion was 325 (213) minutes. The peak creatine kinase concentration was 3737 (2074) IU/l. Twenty six patients (90%) developed Q wave infarction. Twelve patients (41%) had a history of hypertension, 7 (24%) had diabetes mellitus, 8 (28%) had a cholesterol concentration  $> 5.7$  mmol/l, and 15 (52%) were smokers. There was no clinical, ECG, or enzymatic evidence of reinfarction in the hospital. In-hospital medications were as follows: nitrate (100%,  $n = 29$ ), angiotensin converting enzyme inhibitor (97%,  $n = 28$ ),  $\beta$  adrenergic blocking agent (21%,  $n = 6$ ), and diuretics (7%,  $n = 2$ ). None of the patients had atrioventricular block, chest pain, flushing, or palpitation during the infusion of adenosine triphosphate in the TTDE study. The time required to perform TTDE was nine minutes (range 6–16 minutes).

### Angiographic results

Primary coronary angioplasty including stent placement was successful in all patients. Rescue stenting was performed in 26 patients (90%). The culprit lesion was the proximal left anterior descending coronary artery in 16 patients (55%). Eleven patients (38%) had multivessel disease. Twenty two patients (76%) showed poor or no collateral flow (Rentrop grade 1 or 0) on the initial coronary angiogram. Percentage diameter stenosis decreased significantly from before to after coronary angioplasty (from 98 (4)% to 16 (14)%;  $p < 0.0001$ ). Minimum lumen diameter increased significantly from before to after coronary angioplasty (from 0.10 (0.13) mm to 2.70 (0.45) mm,  $p < 0.0001$ ). Three patients (10%) showed TIMI grade 2 reflow, and the remaining 26 patients (90%) showed TIMI grade 3 reflow. None of the patients had significant stenosis distal to the culprit lesion and none had significant restenosis at predischarge.

### Regional left ventricular wall motion and CFVR

A-WMSI decreased significantly from baseline to discharge (from 2.59 (0.16) to 1.93 (0.45),  $p < 0.0001$ ) and the change

**Figure 2** Relation between coronary flow velocity reserve (CFVR) 24 hours after revascularisation and the anterior wall motion score index (A-WMSI) at discharge.

in A-WMSI was 0.64 (0.44). CFVR increased from immediately to 24 hours after revascularisation (from 1.42 (0.09) to 1.54 (0.30),  $p = 0.02$ ) and to discharge (2.13 (0.39),  $p < 0.00001$  v immediately and 24 hours after revascularisation).

### Relation between CFVR and regional wall motion of the infarct zone

On the basis of the results of recent studies, we defined viable myocardium as A-WMSI of 2.00 at discharge.<sup>11</sup> We divided our patients into two groups: those with viable myocardium (A-WMSI at discharge of 2.00,  $n = 18$ ) and those with non-viable myocardium (A-WMSI at discharge  $> 2.00$ ,  $n = 11$ ).

The viable myocardium group had a higher rate of TIMI grade 3 reflow and a lower peak creatine kinase than the non-viable myocardium group. However, there were no significant differences in baseline characteristics, in-hospital medications, or residual stenosis (table 1). With regard to A-WMSI by two dimensional echocardiography, the viable myocardium group had a lower A-WMSI at discharge than the non-viable myocardium group; however, there was no significant difference in A-WMSI before revascularisation (table 2). With regard to CFVR by TTDE, the viable myocardium group had a higher CFVR immediately and 24 hours after revascularisation and at discharge than the non-viable myocardium group (table 2). CFVR immediately and 24 hours after revascularisation were compared with A-WMSI at discharge. CFVR immediately and 24 hours after revascularisation significantly correlated with A-WMSI at discharge (table 3). By a receiver operating characteristic curve analysis,<sup>12</sup> an optimal cut off ratio of 1.5 for CFVR 24 hours after revascularisation was chosen to predict viable myocardium (sensitivity = 94%, specificity = 91%) (fig 2).

### Interobserver and intraobserver variability

The mean percentages of interobserver and intraobserver variability for the measurement of CFVR were 4.0% and 2.8%, respectively. The interobserver and intraobserver variations were within acceptable ranges (interobserver difference 0.05,  $r = 0.93$ ,  $y = 0.98x - 0.01$ ,  $p < 0.0001$ ; intraobserver difference 0.03,  $r = 0.96$ ,  $y = 0.90x + 0.10$ ,  $p < 0.0001$ ).



## DISCUSSION

In this study, we measured coronary blood flow velocity after successful revascularisation by TTDE in patients with acute myocardial infarction and investigated the relation between CFVR and the recovery of regional left ventricular function at the late stage. The main findings were:

- CFVR after revascularisation predicted the recovery of regional left ventricular function
- CFVR 24 hours after revascularisation was a better predictor of recovery of regional left ventricular function than CFVR immediately after revascularisation
- The group with viable myocardium had a higher CFVR immediately and 24 hours after revascularisation and at discharge than the group with non-viable myocardium.

CFVR measured by invasive methods is an established tool for assessing microvascular function.<sup>13</sup> Recent studies have shown that CFVR of the infarct related artery is severely impaired immediately after revascularisation but that it subsequently recovers.<sup>14–15</sup> Improvement of CFVR after adequate reperfusion may be explained by a greater availability of vasodilating mediator substances after the depletion of such substances during ischaemia.<sup>16</sup> Previous studies have shown that CFVR immediately after revascularisation, as determined by invasive methods in patients with acute myocardial infarction, predicts the recovery of regional left ventricular function and that the degree of microvascular injury immediately after revascularisation may be an important factor in predicting the recovery of regional left ventricular function.<sup>4</sup> These findings agree with the present results. Previous animal studies have shown that no reflow associated with microvascular occlusion may continue for several hours after reperfusion and that an initial decrease in CFVR after reperfusion may be followed by a period of recovery that lasts up to one week.<sup>16–18</sup> The degree of the progression of microvascular injury by perivascular oedema, capillary leucocyte plugging, and damage to the vascular endothelium after myocardial ischaemia may hinder the recovery of regional left ventricular function. A recent study showed that CFVR in patients with reperfusion injury as judged by ECG criteria was lower than that in patients without reperfusion injury.<sup>19</sup>

Furthermore, in this study, CFVR 24 hours after revascularisation was a better predictor of recovery of regional left ventricular function than CFVR immediately after revascularisation. Microvascular function may change with time, particularly immediately after reperfusion, and microcirculation may be modified by different factors. This phenomenon has been explained by various mechanisms: microvascular stunning where the microcirculation cannot increase coronary flow velocity or reactive hyperaemia where high postischaemic baseline flow velocities mask an actual CFVR. These mechanisms may hinder the measurement of actual CFVR immediately after revascularisation and thus lead to an underestimation of CFVR.

CFVR can be measured by invasive methods only in the catheterisation laboratory, which precludes long term continuous measurement. However, TTDE, which is non-invasive, can be used to measure CFVR frequently and in the long term in the clinical setting.<sup>5–6</sup> These are some advantages of measuring CFVR by TTDE; in this study we measured CFVR three times (immediately and 24 hours after coronary angioplasty and at discharge). In the future, the measurement of CFVR by TTDE may be useful for evaluating the time course of CFVR. CFVR (especially CFVR 24 hours after revascularisation) by TTDE was useful for predicting the recovery of left ventricular function in patients with acute myocardial infarction.

Recent studies have reported that dobutamine stress echocardiography and myocardial contrast echocardiography were useful for predicting left ventricular wall motion recovery in

patients with acute myocardial infarction.<sup>20–22</sup> However, we performed this study about three years ago, at a time when there was no contrast agent available for intravenous myocardial contrast echocardiography in the acute phase of acute myocardial infarction. Furthermore, intracoronary myocardial contrast echocardiography can be used only in the catheterisation laboratory and has been difficult to apply in routine clinical practice.

In the present study, the measurement of CFVR by TTDE was safe, inexpensive, highly reproducible, and easy. All of these points are beneficial when compared with results obtainable by dobutamine stress echocardiography. However, the measurement of CFVR by TTDE may not be the best approach for predicting left ventricular wall motion recovery in patients with acute myocardial infarction. A future study should be conducted to compare dobutamine stress echocardiography and myocardial contrast echocardiography with the measurement of CFVR by TTDE.

## Study limitations

Firstly, we could not obtain satisfactory coronary blood flow velocity profiles by TTDE in 15% of the patients (5 of 34), and this procedure was performed only for the left anterior descending coronary artery. Secondly, we assumed that A-WMSI at discharge was an indicator of myocardial viability. While this parameter is considered to be indicative of myocardial viability, it is semiquantitative. These results should be compared with those of a single photon emission computed tomography or positron emission tomography study to assess the degree of myocardial damage more quantitatively. Thirdly, we did not consider the influence of diabetes mellitus or other coronary risk factors for vasomotor tone, since the sample size in this investigation was too small. Fourthly, since CFVR may vary with residual stenosis of the epicardial coronary artery, the effect of stenosis needs to be minimised by adequately reducing residual stenosis. In this study, coronary angioplasty was performed as rescue stenting in 90% of the patients and none of the patients had significant restenosis at the predischARGE angiogram. However, if residual stenosis was not detected in measurements of CFVR by TTDE, acute recoil may have occurred. Lastly, angle correction was needed in each examination and this may underestimate or overestimate the true velocity. However, since CFVR is a ratio of two velocities, angle correction should not influence CFVR.

## Clinical implications

In the present study, we showed that CFVR as determined by TTDE after revascularisation in patients with acute myocardial infarction predicted the recovery of regional left ventricular function. This should be helpful for treating patients with reperfused acute myocardial infarction.

## Authors' affiliations

**Y Ueno, Y Nakamura, M Kinoshita**, The First Department of Internal Medicine, Shiga University of Medical Science, Otsu, Japan  
**T Fujita, T Sakamoto, H Okamura**, Okamura Memorial Hospital, Shimizu, Japan

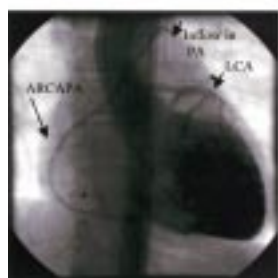
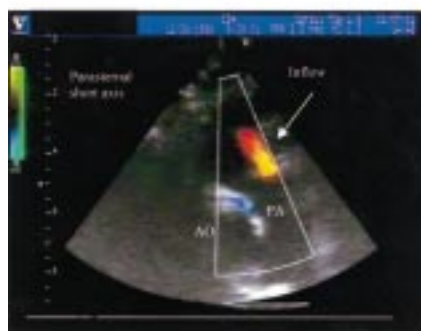
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## IMAGES IN CARDIOLOGY

### A rare congenital coronary anomaly: anomalous origin of the right coronary artery from the pulmonary artery



Whereas anomalous origin of the left coronary artery from the pulmonary artery is rather well known (Bland-White-Garland syndrome, ALCAPA), only a few cases have been described with anomalous origin of the right coronary artery from the pulmonary artery (ARCAPA).

A 12 year old patient was referred with this provisional diagnosis. He had been followed since the age of 5 years because of a muscular ventricular septal defect which had closed spontaneously. There were no pathological signs at physical examination; exercise tolerance was normal. The ECG showed incomplete right bundle branch block without signs of ischaemia. Echo revealed trivial tricuspid and pulmonary valve regurgitation with normal right ventricular pressure. The left coronary artery (LCA) appeared with a large central part of the left anterior descending artery (LAD) but without a visible circumflex artery. The right coronary (diameter 3 mm) was visible anterior to the aortic root (AO) arising from the pulmonary artery (PA) (top panel). There was a “retrograde” laminar blood flow into the PA (middle panel). Angiography confirmed the normal course of the left coronary artery with delayed retrograde visualisation of the right coronary artery via collaterals and run-off of the contrast medium into the pulmonary artery (bottom panel).

Although there are no signs of myocardial ischaemia orthotopic direct reimplantation of the anomalous right coronary artery is planned. The variable and not precisely predictable course of Bland-White-Garland syndrome were taken into consideration for this decision.

**C Rotzsch**  
**M Kabus**  
**H-J Häusler**

rotc@medizin.uni-leipzig.de